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FEATURES OF CHRONIC HEPATITIS IN THE CLINIC OF INTERNAL DISEASES (Literature review)

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Resume. Liver diseases, such as chronic alcoholic hepatitis, chronic hepatitis of viral etiology, cirrhosis of the liver, autoimmune liver pathology, have become widespread among the Asian population. This is due to the low efficiency of modern methods of treatment of chronic hepatitis, as well as the low knowledge of the molecular mechanisms of these diseases, which makes it difficult to conduct pathogenetic therapy.

ОСОБЕННОСТИ ХРОНИЧЕСКИХ ГЕПАТИТОВ В КЛИНИКЕ ВНУТРЕННИХ БОЛЕЗНЕЙ (Обзор литературы)

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Резюме. Заболевания печени, такие как хронический алкогольный гепатит, хронический гепатит вирусной этиологии, цирроз печени, аутоиммунная патология печени, получили широкое распространение среди населения Азии. Это обусловлено низкой эффективностью современных методов лечения хронических гепатитов, а также малой изученностью молекулярных механизмов этих заболеваний, что затрудняет проведение патогенетической терапии.

Of the many problems concerning the immune system of the liver, we have identified several areas that are most significant from our point of view. One of the directions of the search is a detailed analysis of changes related to the immune status of patients with chronic hepatitis. Accurate data on the lymphocyte subpopulation in the liver allows targeted removal of the most aggressive subpopulations causing damage to liver tissue.

The term "chronic hepatitis" has been known since the 30s of the 20th century, and its nosological independence was determined in the 60s. Chronic hepatitis is a polyetiological persistent liver injury lasting at least 6 months and accompanied by an increase in the activity of transaminases and other enzymes that are markers of inflammation [9].

The classification of chronic hepatitis is constantly being improved.

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The classification includes an assessment of the activity of the process (minimal, mild, moderate and pronounced), as well as the stages of the disease by the severity of fibrosis (I – mild fibrosis, II – moderate fibrosis, III – severe fibrosis). In our country, according to the degree of activity, chronic hepatitis is divided into persistent, active and lobular.

Chronic persistent hepatitis is characterized by the expansion of portal tracts and portal inflammatory cell infiltration. Lobular architecture is preserved, fibrosis is absent or weakly expressed. In chronic active hepatitis, the inflammatory infiltrate spreads further to the liver parenchyma with a violation of the integrity of the border plate, there are facies necrosis, intra-lobular septa, the architecture of the lobules is broken, but there is no nodal regeneration of the parenchyma [19].

Foreign classifications.

According to morphological changes, liver diseases are classified as parenchymal, hepatobiliary and vascular [2].

Parenchymal lesions include hepatitis, cirrhosis, infiltrative liver lesions, bulky formations and functional disorders accompanied by jaundice. Cirrhosis of the liver is considered as a separate disease related to chronic degenerative liver damage, in which blood circulation in the liver can be significantly disrupted, which can lead to portal hypertension due to reverse blood flow. Hepatitis is divided into viral, medicinal, toxic, ischemic, which can be acute, subacute and chronic. On the etiological basis, chronic hepatitis is considered as viral and non-viral.

Histological characteristics of a normal liver.

The microarchitectonics of the liver is not homogeneous, it is characterized by the presence of a variable composition of hexagonal and pentagonal lobular units containing traditional portal triads. Liver acinuses are included in lobular structures as smaller triangular physiological units. The variability of liver architectonics is more pronounced on the periphery of the organ.

Numerical equality of hepatic arteries and bile ducts is an indicator of the safety of the biliary system of the liver. A decrease in the diameter of the bile ducts compared to the diameter of the hepatic arteries indicates the presence of destructive processes in the bile ducts, and their expansion indicates hyperplasia.

Nonspecific reactive hepatitis.

Nonspecific reactive hepatitis is a secondary hepatitis caused by a number of endogenous and exogenous factors, reflecting the reaction of the liver to any somatic disease. It is characterized by moderately pronounced morphological changes in the liver, moderately pronounced clinical and laboratory parameters and a benign course. Nonspecific reactive hepatitis can be detected as the initial clinical and morphological stage of liver damage under the influence of exogenous factors, including drugs, drugs and toxins.

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The pathogenesis of the disease is associated with a violation of the detoxification function of the liver in relation to antigens and toxins entering the liver through the portal vein or hepatic artery.

Depending on the localization of inflammatory changes in the liver, lobular and portal variants are distinguished, and according to the degree of activity – persistent and active.

Lobular nonspecific hepatitis is characterized by focal and drain necrosis of the parenchyma, which are located around the central vein. In areas of necrosis, the argyrophilic stroma is destroyed, clusters of macrophages, lymphocytes and neutrophils are visible. Portal tracts are edematous, focal or diffuse sclerosis is noted.

Portal nonspecific hepatitis is characterized mainly by edema and expansion of the portal tracts, histiocytic infiltration and a certain number of lymphocytes. Hepatocytes are in a state of protein and fat dystrophy, necrosis of individual hepatocytes is noted.

In the early stages of fibrosis, the process extends only to the portal areas. At the 2nd stage, the process extends to the periportal region. At the 3rd stage, the process affects the central area, bridge-like fibrosis forms towards the portal or central area. The second and third stages are intermediate, whereas the 4th stage is cirrhosis, in which fibrosis alternates with elements of regeneration, which causes a change in the architectonics of the liver.

Chronic alcoholic hepatitis.

Depending on the clinical picture and duration of the course of the disease, chronic alcoholic hepatitis is divided into stages of inflammation, steatosis and cirrhosis. Alcoholic hepatitis is characterized by a fairly early violation of the structure of the lobules and the transition of the disease to the stage of cirrhosis.

Characteristic morphological differences between alcoholic cirrhosis of the liver and primary biliary cirrhosis is the predominant occurrence of fibrosis around the central veins, which culminates in the formation of small nodules. Whereas in primary biliary cirrhosis, the destruction of the bile ducts initially occurs due to the death of epithelial cells, which is accompanied by fibrosis of the tissue of the portal tracts.

In chronic alcoholic hepatitis, the number of fat droplets in the liver stellate cells increases, the endoplasmic reticulum hypertrophs, the cells begin to synthesize cytokines that promote fibrogenesis. As a result, collagen fibers accumulate in the Disse space, and circulation between cells is disrupted.

With chronic alcoholic hepatitis in the active stage, the number of lymphoid follicles increases, in the center of which there are clusters of macrophages, sometimes containing hemosiderin. However, portal hemosiderosis (that is, the presence of hemosiderin granules in macrophages infiltrating portal tracts) is characteristic of

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medicinal hepatitis, which indicates a common clinical and morphological manifestations of alcoholic and medicinal hepatitis.

Fatty hepatosis.

Fatty infiltration, fatty degeneration, hepatic steatosis or fatty hepatosis are synonyms of one disease that has characteristic clinical signs and is morphologically characterized by the deposition of neutral lipids in hepatocytes and in the extracellular space. If fatty degeneration is combined with hepatitis, it takes a back seat.

Fatty liver dystrophy can have various etiologies, including obesity, starvation, endocrine diseases, a condition after surgery on the gastrointestinal tract, viral infections, toxic liver lesions. allergic diseases, pregnancy pathology, and also be a stage of development of chronic alcoholic hepatitis. Toxic substances that cause the onset of fatty liver dystrophy can also lead to the development of necrotic and inflammatory changes in hepatocytes. With high activity of the process, the degree of fibrosis of liver tissue and the transition of the disease to the stage of necrosis is increased.

Cirrhosis of the liver.

Chronic hepatitis can have different etiologies and mechanisms of liver tissue damage, however, the end result is reduced to progressive fibrosis and cirrhosis.

The pathognomonic morphological picture of liver cirrhosis is diffuse hyperplasia of liver tissue, expressed in the development of parenchymal nodules surrounded by fibrous septa connecting the portal canal and the central one, and accompanied by a restructuring of vascular architectonics. The mechanism that causes the development of parenchymal nodes is the activation of hepatocyte growth after necrotic death of parenchymal cells, the separation of lobules into parts by fibrous strands, the change in the structure of lobules due to the restructuring of the vascular bed. One of the molecular mechanisms of the development of liver cirrhosis is a violation of the activation and differentiation of liver stem cells, which will be discussed further.

Cirrhosis is characterized by loss of functional mass of the liver and functional insufficiency, increasing with the progression of the disease. The early stages of the disease enable the liver to compensate for the loss of cell mass. Then comes a period of decompensation, accompanied by a decrease in the number of cells per unit volume of the liver, which ends with the death of the body. This period can last from 1-2 years to 20-30 years.

Autoimmune damage to the liver parenchyma.

Autoimmune reactions are characterized by the production of antibodies that react with the structural components of their own cells and tissues. With autoimmunity, effector T-lymphocytes are formed that recognize their own peptides. Activation of T-lymphocytes occurs both nonspecifically and cross-reacting antibodies. Autoimmunity

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is divided into systemic and organ-specific. Autoimmune liver lesions include primary biliary cirrhosis, sclerosing cholangiitis, autoimmune chronic hepatitis.

In the liver, there is a loss of tolerance to its own antigens, which can begin without apparent clinical reasons. However, such patients almost always had a history of some kind of viral infection. Viral infections, including the influenza virus, can serve as a trigger for the development of autoimmunity. The mechanism of initiation of autoimmune reactions by viruses can be the production of so-called antigen-dependent nonspecific immunoglobulins, the production of which goes in parallel with the synthesis of antibodies, but exceeds them by an order of magnitude. In patients with primary cirrhosis of the liver, the level of immunoglobulins of all classes is increased, which indicates polyclonal activation of lymphocytes.

Recently, the issue of the genetic predisposition of some individuals to autoimmune liver pathology has been actively discussed. For example, antimitochondrial antibodies, which are found in 95% of patients with primary biliary cirrhosis, are also found in practically healthy individuals. Dispensary observation of these patients showed that sooner or later they begin to develop liver damage [7].

Literature

1. Нурмагомаев М.С., Магомедова З.С., Каграманова З.С. ЛИТЕРАТУРНЫЙ ОБЗОР: ХРОНИЧЕСКИЕ ГЕПАТИТЫ В КЛИНИКЕ ВНУТРЕННИХ БОЛЕЗНЕЙ // Научное обозрение. Медицинские науки. – 2016. – № 5. – С. 77-91; URL: https://science-medicine.ru/ru/article/view?id=932 (дата обращения: 21.08.2023).
2. Хидоятов Б.А., Шокиров Б.С., Садыкова Э.И., Эргашев Н.А. Морфологические особенности тонкой кишки при экспериментальном сахарном диабете// Журнал “Морфология”. - 2016. - №4. – С. 219.Cookson W.O.C.M., Sharp P.A. et al. Lancet 1989; 1: 1292-5.
3. БС Шокиров. (2019). ВЛИЯНИЕ ПРОТИВОДИАБЕТИЧЕСКИХ ПРЕПАРАТОВ СИОФОР И ГЛИКЛАЗИД НА МОРФОЛОГИЧЕСКИЕ ОСОБЕННОСТИ ТОЛСТОГО КИШЕЧНИКА ПРИ ЭКСПЕРИМЕНТАЛЬНОМ САХАРНОМ ДИАБЕТЕ// НЕДЕЛЯ НАУКИ – 2019, 832-834.
4. Uehara M., Kimura C. Acta Derm.Venerol.Suppl.(Stockh.), 1993; 73: 62–3.
5. Williams H.S., Burney P.G.J., Pembroke A.C. et al. Br. J. Dermatol. 1993; 131: 406–16.
6. Halimova Yu.S. Shokirov B.S. Morphological changes of internal organs in chronic alcoholism// Middle European Scientific Bulletin 12, 51-55.
7. Hide D.W., Arshad S.H., et al. Clin.Exp.Allergy 1991; 21: 739–43.
8. Ю.С.Халимова. (2021). MORPHOFUNCTIONAL ASPECTS OF THE HUMAN BODY IN THE ABUSE OF ENERGY DRINKS// Новый день в медицине. 5 (37), С. 208-210.
9. Б.С Шокиров. (2021). MORPHOLOGICAL FEATURES OF THE LYMPHOID TISSUE OF THE SMALL INTESTINE IN AN ADULT// Новый день в медицине. 5 (37), С. 240-242.